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Chapter 63: Supportive Care in Cancer

INTRODUCTION

- Anticancer treatments often cause adverse drug reactions (ADRs), many of which can be life-threatening if not appropriately managed. These toxicities often result from the agent's mechanism of action or "off-target" effects.
- Cytotoxic chemotherapy targets rapidly dividing cells, often causing myelosuppression, gastrointestinal (GI) effects (mucositis, diarrhea, etc), alopecia, and infertility.
- Biologic therapies and targeted small molecule agents cause various ADRs based on altered intracellular signaling, for example, rash with epidermal growth factor receptor (EGFR) inhibitors and hemorrhage or thrombosis with vascular endothelial growth factor (VEGF) inhibitors. Immunotherapies have a unique ADR profile, including autoimmune toxicities from immune system modulation.
- Toxicities are commonly graded using the Common Terminology Criteria for Adverse Events (CTCAE) scale from 0 (no toxicity) to 5 (death). Strategies for preventing and managing ADRs associated with anticancer therapies are detailed below.

HEMATOLOGIC TOXICITIES: ANEMIA, NEUTROPENIA, AND THROMBOCYTOPENIA

- Normal hematopoiesis involves several coordinated steps. Pluripotent stem cells differentiate, proliferate, and mature into blood cells found in peripheral circulation. Myeloid stem cells form erythrocytes, platelets, monocytes, basophils, neutrophils, and eosinophils. Lymphoid stem cells form B-lymphocytes, T-lymphocytes, and natural killer cells.
- Myelosuppression (depressed bone marrow function causing anemia, neutropenia, and thrombocytopenia) can result from cancer in the bone marrow or anticancer treatments. Some suppression is acceptable if quality of life is not compromised and the cancer is responding. When cure is the goal, hematopoietic growth factors may be considered as an alternative to dose reduction.

NEUTROPENIA

- Neutropenia increases infection risk, especially when the absolute neutrophil count is <500 cells/mm^3 (0.5×10^9/L) or lasts >7–10 days. Other risk factors for infection include compromised physical barriers and impaired leukocyte function, which may result from the cancer itself, anticancer agents, or radiation therapy (RT).
- Infections are difficult to identify in neutropenic patients because typical signs and symptoms (eg, purulent drainage) are often absent. Clinicians therefore rely on fever as a key indicator. Prompt initiation of empiric antibiotics, guided by patient history and presentation and local antibiograms, is critical to prevent mortality. Most infections originate from the patient's own flora, including gram-positive and gram-negative bacteria.
- **Management**

  ✓ Colony-stimulating factors (CSFs) may reduce neutropenia severity and infection risk by providing proteins essential for blood cell growth and maturation. Start daily **filgrastim** or single-dose **pegfilgrastim** 24–72 hours postchemotherapy. CSFs are generally well tolerated; bone pain is the most common ADR. Consult guidelines for appropriate CSF use based on a regimen's risk of causing febrile neutropenia (FN).

  ✓ Primary prophylaxis, the prevention of neutropenia with the first cycle of chemotherapy, is recommended with regimens when the risk of FN is ≥20%. Most dose-dense regimens require CSF support to maintain dose intensity and schedule.

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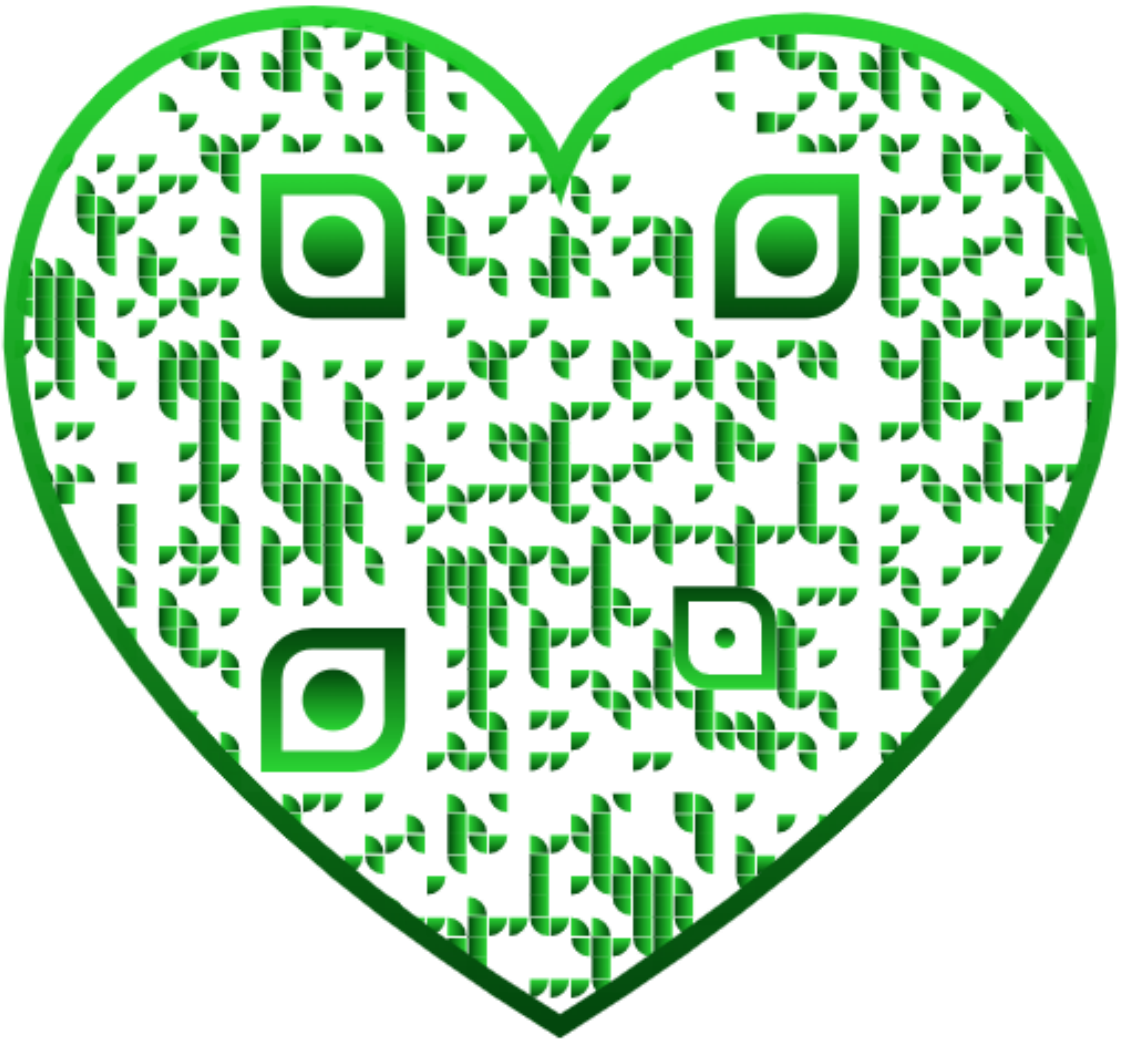

✓ For intermediate FN risk (10%–20%), consider primary prophylaxis if one or more risk factors are present: age ≥65 years receiving full chemotherapy dose intensity, prior chemotherapy or RT, persistent neutropenia, bone marrow involvement, recent surgery and/or open wounds, liver or renal dysfunction.

✓ Consider secondary prophylaxis in patients who developed FN with the prior cycle of chemotherapy when the cancer is chemosensitive and dose reduction or delay could impact survival.

✓ **Trilaciclib,** a first-in-class myeloprotective agent, is approved in extensive-stage small cell lung cancer and administered intravenously on each day of chemotherapy. Common ADRs include fatigue, hepatotoxicity, and electrolyte imbalances. Avoid use with cisplatin due to increased risk of nephrotoxicity.

ANEMIA

- Anemia is a common complication after cytotoxic chemotherapy and with various tyrosine kinase inhibitors (TKIs), such as those targeting EGFR, human epidermal growth factor receptor 2 (HER2), and VEGF.
- **Management**

  ✓ Red blood cell transfusion is the mainstay of treatment.

  ✓ Consider erythropoiesis-stimulating agents (ESAs; **epoetin alfa**, **darbepoetin alfa**) in patients with kidney disease or on palliative care with hemoglobin <10 g/dL (100 g/L; 6.21 mmol/L).

  ✓ ESA use is limited due to serious ADRs, including thrombosis, myocardial infarction, and increased risk of death in cancer patients.

THROMBOCYTOPENIA

- Thrombocytopenia associated with chemotherapy increases the risk for significant bleeding.
- **Management**

  ✓ Platelet transfusion is the mainstay of treatment. Consider a transfusion when platelet count <10,000 cells/mm^3 (10×10^9/L) or higher if the patient is experiencing active bleeding, undergoing an invasive procedure, has coagulation abnormalities (eg, acute promyelocytic leukemia), or has fever/documented infection.

GI TOXICITIES: DIARRHEA, MUCOSITIS, AND NAUSEA/VOMITING

DIARRHEA

- Mucosal damage can occur at any part of the GI tract. In the lower tract, it usually presents as diarrhea and abdominal pain, with symptom onset varying by agent (eg, within days for cytotoxic chemotherapy versus delayed after immunotherapy). Monitor daily stool count, electrolytes, and fluid balance since supplementation may be necessary.
- Anticancer agents known to commonly cause diarrhea include irinotecan, sacituzumab govitecan, 5-fluorouracil, capecitabine, and targeted therapies (eg, imatinib, EGFR, HER2, VEGF, phosphoinositide-3 kinase [PI3K], cyclin-dependent kinase, or cyclin dependent kinase 4/6 inhibitors) and immune checkpoint inhibitors (ICIs).
- **Management**

  ✓ Rule out infectious causes.

  ✓ Treat mild-to-moderate diarrhea with **loperamide** (consider 4-mg dose orally [PO], then up to 2 mg PO every 2 hours). Use **octreotide** for persistent or severe diarrhea. **Diphenoxylate/atropine** (diphenoxylate 5 mg PO four times daily; maximum 20 mg/day) may be added.

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✓ Nonpharmacologic interventions include cessation of lactose, alcohol, or high-osmolar supplements; encouraging clear liquids and small frequent meals; and considering the BRAT diet (banana, rice, applesauce, toast).

✓ Severe or complicated diarrhea (eg, dehydration, hypotension, bleeding, fever, neutropenia, severe cramping, unresponsive to intervention) warrants hospital admission with hydration, antibiotics, and octreotide. Anticancer treatment is often temporarily held.

MUCOSITIS

- GI mucosa is a common site of toxicity from anticancer therapy, particularly cytotoxic agents (eg, fluorouracil, methotrexate) and targeted therapies like multikinase and mammalian target of rapamycin (mTOR) inhibitors. Inflammation (mucositis) can cause painful ulcerations and impair eating, drinking, or swallowing. Disruption of the GI barrier also increases the risk of systemic microbial invasion and infection.
- Mucositis prevention relies on good oral hygiene. Oral cryotherapy is recommended before high-dose melphalan and 5-fluorouracil to reduce drug delivery through vasoconstriction. **Palifermin,** a keratinocyte growth factor, may help patients undergoing specific hematopoietic stem cell transplant (HSCT) regimens. Dexamethasone mouthwash is effective for mTOR-associated mucositis.
- **Management**

  ✓ Treatment is mainly supportive, including use of topical and systemic analgesics.

  ✓ Mild infections (ie, thrush) may be treated with topical antifungals, such as **clotrimazole troches** or **nystatin oral suspension**, whereas severe oral or esophageal fungal infections require systemic antifungals. See Chapter 37 for more information on treatment of superficial fungal infections.

  ✓ Severe mucositis can cause dehydration, requiring intravenous (IV) fluids and opioid analgesics; prolonged cases may need IV nutrition. Culture suspicious lesions and initiate appropriate antifungal or antiviral therapy. See Chapter 36 for more information on treatment of invasive fungal infections.

NAUSEA/VOMITING

- Chemotherapy-induced nausea and vomiting (CINV) is a physiologic and psychological process that significantly impacts quality of life. CINV may cause metabolic derangements, nutritional depletion, anorexia, weight loss, early cessation of anticancer treatment, and reduced self-care or functional ability.
- Antiemetic prophylaxis is based on emetogenicity—the potential for an agent to cause nausea/vomiting (N/V). Guidelines classify IV therapies into four risk categories: highly (HEC), moderately (MEC), low (LEC), and minimally emetogenic chemotherapy corresponding to CINV rates of >90%, 30%–90%, 10%–30%, and <10%, respectively. *Oral* anticancer therapy is classified as minimal-to-low (<30%) or moderate-to-high (≥30%) emetogenic risk. See Chapter 27 for more information on nausea and vomiting.
- **Management**

  ✓ Antiemetic prophylaxis is based on risk: HEC, quadruplet (or triplet, per National Comprehensive Cancer Network [NCCN]); MEC, doublet (or triplet, per NCCN); LEC, monotherapy; minimal, none (**Table 63-1**). The goal is complete prevention of nausea and vomiting during the risk period. Emetic risk lasts 2 days for MEC and 3 days for HEC. Antiemetics should start before any regimen with ≥10% risk, generally 30 minutes prior, and continue through the risk period.

  ✓ For *oral anticancer* therapy prophylaxis, consider the setting (inpatient vs outpatient), administration route(s), overlap with IV chemotherapy, risk duration, duration of antiemetic efficacy, tolerability of prolonged use, and adherence.

  ✓ *Radiation-induced* N/V prevention is based on emetogenic risk, which depends on RT site. Optimal medication, dose, and duration of prophylaxis are unknown. If receiving concomitant RT and chemotherapy, base prophylaxis on the higher emetogenic risk.

  ✓ *Anticipatory N/V* is often a result of prior negative experiences—patient education is key. **Lorazepam** is commonly used.

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✓ Provide breakthrough (ie, as needed) medication at symptom onset. Select agent based on prior prophylaxis and potential ADRs. Options include **olanzapine, phenothiazines**, **metoclopramide**, **dronabinol, scopolamine patch**, **haloperidol, 5-hydroxytryptamine/serotonin receptor antagonists**, **dexamethasone,** or **benzodiazepines**.

TABLE 63-1

CINV Prophylaxis for IV Chemotherapy Regimens

CINV Prophylaxis Regimen Definitions			
Quadruplet Therapy	**Triple Therapy**	**Doublet Therapy**	**Monotherapy**
5-HT3-RA + steroid + NK1-RA + olanzapine	5-HT3-RA + steroid + NK1-RA *or* olanzapine	5-HT3-RA + steroid	5-HT3-RA *or* dexamethasone (per ASCO) *or* metoclopramide *or* prochlorperazine (per NCCN) All ONCE doses

CINV Prophylaxis Recommendations for IV Chemotherapy		
Risk	**Phase**	**Prophylaxis**
HEC	Acute phase	Quadruplet: 5-HT3-RA + dexamethasone + NK1-RA + olanzapine (can utilize triplet therapy)
	Delayed phase	Olanzapine on days 2–4 If used on day 1, oral aprepitant continues days 2–3 If regimen other than AC, dexamethasone continues days 2–3 (dose varies by which NK1 utilized)
MEC	Acute phase	Doublet[a]: 5-HT3-RA + dexamethasone
	Delayed phase	Dexamethasone 8 mg PO/IV on days 2–3 only if patients receiving therapies with known potential for delayed CINV
LEC	Acute phase	Monotherapy: 5-HT3-RA or dexamethasone
	Delayed phase	None
Minimal	Acute phase	None
	Delayed phase	None

5-HT3-RA, 5-hydroxytryptamine/serotonin receptor antagonist; AC, anthracycline + cyclophosphamide; ASCO, American Society of Clinical Oncology; NK1-RA, neurokinin-1 receptor antagonist.

Note: Per ASCO guideline, carboplatin with AUC ≥4 specifically warrants prophylaxis in acute phase with 5-HT3-RA + dexamethasone + NK1-RA and none in delayed phase. When given at equipotent doses, oral and IV serotonin receptor antagonists are equivalent in efficacy.

[a]Optional: triplet regimen based on patient-specific risk factors (ie, NK1-RA- or olanzapine-based triplet).

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ONCOLOGIC EMERGENCIES: TUMOR LYSIS SYNDROME (TLS) AND EXTRAVASATION

- Oncologic emergencies are acute complications associated with cancer pathophysiology and/or the initiation of anticancer treatments. Prompt recognition and initial management are critical to reduce morbidity and mortality. Examples include TLS, extravasation, hypersensitivity reaction, FN, hypercalcemia of malignancy, malignant spinal cord compression, superior vena cava syndrome, malignant pericardial effusion, malignant airway obstruction, disseminated intravascular coagulopathy, hyponatremia, hyperviscosity syndrome, and leukocytosis.

- **TLS**

  ✓ TLS is the most common oncologic emergency caused by rapid cell lysis releasing intracellular contents. Nucleic acid breakdown leads to hyperuricemia, hyperkalemia, hyperphosphatemia, and secondary hypocalcemia.

  ✓ It occurs most commonly in aggressive hematologic malignancies like high-grade lymphomas or acute leukemias. Incidence varies by cancer type and treatment. TLS may follow RT, oral chemotherapy, or targeted therapy (eg, monoclonal antibodies) and typically arises 12–72 hours after treatment starts but can occur earlier or later.

  ✓ TLS monitoring includes labs (uric acid, potassium, calcium, phosphorus), renal function, urine output, fluid status, and, if needed, electrocardiogram (ECG) for arrhythmias and electroencephalogram (EEG) for seizure activity. Prevention and management overlap, so all patients require ongoing monitoring and hydration (**Table 63-2**).

- **Extravasation**

  ✓ Extravasation is the unintentional leakage of a drug into the surrounding subcutaneous or subdermal tissue, potentially causing cell death. Signs and symptoms include pain, redness, swelling, and IV line dysfunction. Incidence is <7%, but it can lead to negative patient outcomes and morbidity.

  ✓ Management depends on the specific chemotherapy agent. Consult product labeling and drug information sources for antidote use, if indicated.

  ✓ Nonpharmacologic interventions include stopping the infusion, aspirating the drug, avoiding line flushing, and elevating the limb for ˜48 hours to promote drainage. Apply warm or cold compresses for 24–48 hours depending on the offending agent. Avoid use of moist compresses and occlusive dressings.

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TABLE 63-2

TLS: Definition, Presentation, and Management

Laboratory TLS[a]	Symptoms/Signs	Management Strategies
Uric acid ≥8 mg/dL (476 µmol/L) or >25% ↑ from baseline	• Obstructive uropathy, hematuria, flank pain, lethargy, oliguria, or anuria	• Prevention: hydration, allopurinol (or potentially febuxostat); consider for low risk and optional for intermediate risk • Management: continue hydration; rasburicase optional for intermediate risk and recommended for high risk; allopurinol for those unable to receive rasburicase
Calcium ≤7 mg/dL (1.75 mmol/L) or >25% ↓ from baseline	• Muscle cramp/spasms, tetany, paresthesia, neurologic (confusion, hallucination, seizure), arrhythmia, bradycardia, cardiac failure, death	• First, manage hyperphosphatemia • If symptomatic, IV calcium may be administered
Potassium ≥6 mEq/L (mmol/L) or >25% ↑ from baseline	• GI (nausea, diarrhea, anorexia), muscle weakness/cramps, ECG abnormalities, sudden death	• Loop diuretic • IV insulin + dextrose • Inhaled albuterol • If mild/moderate—oral resin binder • If ECG changes—IV calcium • If acidotic—IV sodium bicarbonate
Phosphorous ≥4.5 mg/dL (1.45 mmol/L) in adults or ≥6.5 mg/dL (2.1 mmol/L) in children (>25% ↑ from baseline)	• GI (nausea, diarrhea), lethargy, seizures	• Reduce phosphorus intake • Phosphorus binders

[a]Two or more of the laboratory changes must be observed within 3 days before or 7 days after cytotoxic therapy.

IMMUNE-RELATED ADVERSE EVENTS

- ICIs activate the immune system but also may attack healthy cells, causing immune-related adverse events (irAEs; incidence 15%–90%). These toxicities may occur during therapy or months to years after discontinuation. irAEs are managed with immunosuppression, typically corticosteroids. Mild-to-moderate cases may allow continued therapy, but corticosteroid use may require holding or stopping ICIs. Dose reduction is not used. irAEs preferentially target skin, gut, endocrine, lungs, and musculoskeletal systems.
- **Management**

  ✓ Rash/dermatitis often appears 2–3 weeks after the first ICI dose; diarrhea/colitis around week 5; hepatotoxicity and hypophysitis around week 7.

  ✓ **Prednisone** (0.5–2 mg/kg/day) is the first-line therapy; severe cases are treated with IV **methylprednisolone** (1–2 mg/kg/day).

  ✓ If no response occurs within 48–72 hours, additional immunosuppression is recommended; options include **tumor necrosis factor**

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inhibitors (ie, **infliximab, etanercept), vedolizumab, mycophenolate**, **IV immunoglobulin**, and **rituximab.**

✓ Consult practice guidelines for up-to-date management recommendations.

CELLULAR IMMUNOTHERAPY ADRs: CRS AND NEUROTOXICITY

- Chimeric antigen receptor T cell (CAR-T) therapy and bispecific antibodies can cause cytokine release syndrome (CRS) and neurotoxicity, requiring close monitoring and prompt intervention with the anti-interleukin-6 agent **tocilizumab** and corticosteroids. Optimal treatment is being evaluated in ongoing studies.

CAR-T THERAPY

- **CRS** usually begins within 7 days of infusion and lasts about 1 week. Monitor vital signs (temperature, blood pressure [BP], and oxygen saturation) twice daily. Grading is based on symptoms.
- **Management**

  ✓ Treat fever with antipyretics; give IV fluids and/or oxygen as needed.

  ✓ For grade ≥2, use tocilizumab 8 mg/kg IV (maximum 800 mg), up to 3 doses in 24 hours. If unresponsive after 1–2 doses, add **dexamethasone** 10 mg IV up to every 6 hours. For refractory grade 4, use **methylprednisolone** 1000 mg/day IV for up to 3 doses; may escalate to every 12 hours.

- **Neurotoxicity (immune effector cell–associated neurotoxicity syndrome)** can occur within 4 weeks postinfusion; duration varies. Signs and symptoms include aphasia, cognitive impairment, reduced level of consciousness, seizures, motor weakness, or cerebral edema.
- **Management**

  ✓ Supportive care with aspiration precautions; if needed, add antiepileptics and manage elevated intracranial pressure.

  ✓ Use dexamethasone 10 mg IV (or methylprednisolone 1000 mg/day IV) for grade 2 or 3. If grade 4, use high-dose steroids such as methylprednisolone with a taper.

BISPECIFIC ANTIBODIES

- CRS and neurotoxicity may occur with these immune-stimulating agents. Additional toxicities include dysgeusia, skin/nail changes, cytopenias, infection, and hepatotoxicity. Hospitalization may be required.

DERMATOLOGIC TOXICITIES

SKIN

- Up to 84% of patients on anticancer therapy experience dry skin and/or pruritus, especially with targeted therapies.
- Acneiform rash, marked by papules and pustules on the face, scalp, upper chest, and back, is common with EGFR and mitogen-activated protein kinase (MEK) inhibitors. Onset is usually within days to weeks of treatment and may include pruritus, stinging, and pain.
- Hand–foot syndrome (HFS), also known as palmar–plantar erythrodysesthesia syndrome, acral erythema, or toxic erythema, is marked by tingling, redness, burning, swelling, and hyperkeratosis of the palms/soles. Lesions may progress to blisters, desquamation, erosions, ulcerations, and bleeding associated with discomfort and moderate-to-severe pain. It is commonly caused by 5-fluorouracil, capecitabine, doxorubicin, pegylated liposomal doxorubicin, docetaxel, and cytarabine. Onset is usually within days to weeks of treatment but may be delayed up to 6 months.
- Hand–foot skin reaction (HFSR) presents with painful, well-defined hyperkeratosis. Multikinase VEGF inhibitors such as sorafenib, cabozantinib,

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and regorafenib are a common cause. It is less commonly seen with lenvatinib, pazopanib, and sunitinib. Grade 3/4 reactions occur in 5%–20% of patients. Onset is usually within days to weeks but may be delayed up to 6 months. Commonly affects the soles with blistering and callus-like hyperkeratosis in pressure-bearing areas.

- Prevention and management strategies are detailed in **Table 63-3**.

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TABLE 63-3

Acneiform Rash/HFS/HFSR

Toxicity	Prevention	Treatment
Acneiform rash (papulopustular exanthema)	• Avoid frequent washing with hot water • Avoid skin irritants, including OTC antiacne medications containing benzoyl peroxide, solvents, or disinfectants • Use alcohol-free[a] OTC moisturizing creams or ointments BID, preferably with urea-containing (5%–10%) moisturizers to the body • Avoid excessive sun exposure • Apply sunscreen (SPF) ≥15 to exposed areas of body and reapply every 2 hours when outside • Oral antibiotics for 6 weeks at start of therapy (doxycycline 100 mg PO BID or minocycline 100 mg PO daily; if unable to tolerate/allergy, cefadroxil 500 mg PO BID or trimethoprim-sulfamethoxazole 160/80 mg PO BID), with or without topical low/moderate-strength steroid to face and chest BID	Grade 1/2: • Continue anticancer treatment • Continue or initiate: oral antibiotic for 6 weeks AND topical low/moderate steroid • Reassess after 2 weeks; if no improvement or worsening escalate therapy Grade ≥3: • Interrupt anticancer treatment until grade 0/1 • Obtain bacterial/viral/fungal cultures if infection is suspected • Continue or initiate: oral antibiotic for 6 weeks AND topical low/moderate steroid, systemic corticosteroids (eg, prednisone 0.5–1 mg/kg × 7 days) • Reassess after 2 weeks; if no improvement or worsening, may need to discontinue treatment
HFS and HFSR	• Avoid irritation to hands and feet; long walks, heavy carrying without protection • Avoid chemical stress; skin irritants, solvents, or disinfectants • Treat predisposing factors before anticancer therapy • Apply 10% urea cream TID • For taxane-based therapy: use skin cooling gloves or socks • For capecitabine-based therapy: may consider celecoxib 200 mg PO BID	• Anticancer treatment interruption and/or dose reduction commonly required; initiation of topical agents (HFS and HFSR) or cooling (HFS) may allow for continued dosing • Hyperkeratosis: treat with keratolytics (topical creams or ointments with 5%–10% salicylic acid or 10%–40% urea) • Inflammation: high-potency topical corticosteroids (eg, clobetasol propionate 0.05%) • Erosions and ulcerations: antiseptic solutions (eg, silver sulfadiazine 1%, polyhexanide 0.02%–0.04%) • Pain: lidocaine 5% cream or patches • Reassess after 2 weeks; if no improvement or worsening, escalate therapy

BID, two times daily; HFS, hand–foot syndrome; HFSR, hand–foot skin reaction; OTC, over the counter; PO, orally; SPF, sun protection factor; TID, three times daily.

[a]Avoid SD alcohol 40, denatured alcohol, ethanol, and isopropyl alcohol.

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HAIR

- Chemotherapy-induced alopecia typically results in diffuse scalp hair loss, although partial or patchy loss can occur. It may also affect eyebrows, eyelashes, and body hair. Hair loss starts 1–3 weeks after treatment; regrowth begins 2–3 months after chemotherapy completion.
- Severity depends on drug type, dose, administration method, and treatment interval. High-risk agents include cyclophosphamide, doxorubicin, irinotecan, and taxanes.
- **Management**

  ✓ Scalp cooling is the only proven preventative method, although it is less effective with breast cancer regimens. After completion of chemotherapy, **topical minoxidil 5%** or oral **biotin** may stimulate hair growth.
- Targeted therapies may cause changes in hair quality, texture, and growth pattern within 2–3 months of treatment.
- **Management**

  ✓ Treatment breaks, antibiotics, corticosteroids, and topical cleansers or barrier agents may be used.

NAILS

- Nail changes, including melanonychia (black or brown discoloration that is diffuse, transverse, or longitudinal), leukonychia (white spots), onycholysis (nail detachment from the nail bed), Beau's lines (indentations across the nail), onychomadesis (proximal separation of the nail plate from the nail matrix), and onychorrhexis (brittle nails) are common during treatment and usually disappear following completion of therapy.
- Preventive strategies include decreased exposure to water, use of cotton gloves, and nail hydration with thick emollients. Consider oral biotin to help with nail growth and use of nail lacquers.

ENDOCRINE TOXICITIES

HYPOTHALAMIC–PITUITARY DYSFUNCTION

- Commonly caused by RT, with onset and severity dose-dependent. Symptoms, which may occur years later, are nonspecific and include fatigue and weakness.
- **Management**

  ✓ Hormone replacement as necessary.

THYROID DYSFUNCTION

- RT, targeted therapy, and cytokines affect thyroid function; hypothyroidism is more common than hyperthyroidism. Neck RT, especially in childhood, is associated with primary hypothyroidism. Approximately half of those receiving RT develop hypothyroidism, with a median onset of 1.5 years posttreatment.
- Monitor thyroid function at baseline and periodically with multitargeted TKIs and ICIs. Hypothyroidism is common with sunitinib, often within 4 weeks of treatment, and may persist following discontinuation. It can also occur with sorafenib, imatinib, dasatinib, nilotinib, and axitinib.
- **Management**

  ✓ Use thyroid replacement hormones as necessary.

  ✓ Patients on thyroid hormones prior to treatment initiation often need dose increases while on TKI treatment. See Chapter 20 for more information on thyroid disorders.

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HYPERGLYCEMIA

- Glucocorticoids, commonly used in patients with cancer, may cause hyperglycemia. They are used as antiemetics, for brain metastasis–related edema, for prevention of transplant rejection, for treatment of graft-versus-host disease and irAEs, and in some anticancer treatment regimens.
- Agents that inhibit the PI3K/protein kinase B (also known as AKT)/mTOR pathway are associated with hyperglycemia, resulting in decreased glucose transport, decreased glycogen synthesis, and increased glycolysis. The PI3K inhibitors (eg, alpelisib) and mTOR inhibitors (eg, everolimus) are associated with grade 3–4 hyperglycemia in approximately 10% of patients.
- **Management**

  ✓ Screen using fasting glucose, oral glucose tolerance test, A1c, and random or pre- or postprandial glucose testing.

  ✓ Consider glycemic variability of the agent administered when considering treatment options (eg, oral antihyperglycemic agents and insulin). See Chapter 19 for more information on diabetes mellitus.

BONE HEALTH

OSTEOPOROSIS

- Osteoporosis is a common complication of anticancer treatment, especially hormonal therapy.
- Consider bone-modifying therapy for high-risk patients, including premenopausal women receiving gonadotropin-releasing hormone therapies or with chemotherapy-induced ovarian failure or prior oophorectomy; postmenopausal women who are receiving aromatase inhibitors; men on or with a history of androgen deprivation therapy; HSCT patients; and those with >3–6 months of glucocorticoid treatment.
- Assessment and treatment is similar to other patients at risk for osteoporosis. See Chapter 3 for more information on osteoporosis.

BONE METASTASES

- Bone metastases commonly affect the axial skeleton causing skeletal-related events (SREs) such as fractures, spinal cord compression, hypercalcemia, and the need for RT or surgery.
- Prevention helps maintain mobility, social function, quality of life, and survival while reducing healthcare costs.
- **Management**

  ✓ Treatment goals include prevention of disease progression and symptom palliation; treatment depends on cancer type.

  ✓ Local beam RT relieves painful bone metastasis with ~1/3 of patients reporting complete pain relief. Radium-223 is used in bone-predominant metastatic castration-resistant prostate cancer.

  ✓ Surgery preserves function and mobility by relieving pain, preventing impending fractures and/or neural compression, or stabilizing a pathological fracture.

  ✓ Bone-modifying agents (BMAs) are potent inhibitors of bone resorption used to relieve pain and reduce SRE risk (see **Table 63-4**).

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TABLE 63-4

BMA Dosing for Bone Metastases

Medication	Brand Name[a]	Dose
Denosumab	Xgeva	120 mg subcutaneously every 4 weeks
Pamidronate	Aredia	90 mg IV over 2 hours every 3–4 weeks
Zoledronic acid	Zometa	4 mg IV over 15 minutes every 3–4 weeks[b]

[a]Note: unique brand names, dosing, and schedule for this indication.

[b]May be dosed every 12 weeks after treatment for 3–6 months.

OTHER TOXICITIES

OCULAR TOXICITIES

Blurred vision, photophobia, conjunctivitis, cataracts, abnormal lacrimation, dry eye, keratitis, optic neuropathy, and retinopathy are commonly reported. They are typically mild but can worsen and lead to blindness.

Management

✓ Follow treatment recommendations for specific complaint.

✓ Baseline assessment and routine eye exams are recommended when using agents associated with serious ocular toxicity.

CARDIOVASCULAR TOXICITIES

- Conduct a thorough cardiac workup before initiating cardiotoxic treatment; any conditions identified must be controlled before treatment.
- Left ventricular dysfunction (LVD)/heart failure is associated with anthracyclines, HER2-targeted therapy, VEGF receptor inhibitors, BRAF/MEK inhibitors, and some proteasome inhibitors (eg, carfilzomib). Anthracycline-induced cardiotoxicity is irreversible (type 1 LVD); risk is cumulative—track lifetime exposure (see **Table 63-5**).
- HER2-targeted agents and VEGF inhibitors cause noncumulative, non–dose-related LVD (type II). Do not give in combination with anthracyclines due to increased cardiovascular risk.
- **Management**

  ✓ Use echocardiogram or multigated acquisition to assess left ventricular ejection fraction and diastolic function prior to cardiotoxic agents, every 3 months during HER2 therapy, after doxorubicin dose of 250 mg/m^2 or equivalent, then after each additional 100 mg/m^2, and posttreatment to evaluate for delayed toxicity.

  ✓ Patients on cardiotoxic agents are considered to have stage A congestive heart failure. See Chapter 11 for more information on heart failure.

  ✓ Consider prophylactic angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or selective beta blockers to decrease the cardiotoxicity risk.

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✓ **Dexrazoxane** may be used as a cardioprotectant in select patients.

- Hypertension (HTN) can occur within 1 day of VEGF inhibitor initiation; it stabilizes within 6–10 days.
- **Management**

  ✓ Monitor resting BP daily during the first cycle and, once stabilized, every 2–3 weeks.

  ✓ Manage per practice guidelines. See Chapter 12 for more information on HTN. Modify HTN management posttherapy to avoid hypotension.

- QTc prolongation may be seen with arsenic trioxide, ivosidenib, vandetanib, crizotinib, vemurafenib, dasatinib, lapatinib, ribociclib, and nilotinib.
- **Management**

  ✓ Correct electrolyte abnormalities prior to initiation and during therapy. Obtain ECG at baseline and periodically to assess for QTc prolongation and arrhythmias.

TABLE 63-5

Lifetime Cumulative Anthracycline Dose Conversion and Maximums

Anthracycline	Conversion Factor	Maximum Recommended Cumulative Dose (mg/m^2)
Doxorubicin	1	550
Daunorubicin	1	550 (no CV risk factors)
	0.7	400 (chest irradiation or CV risk factors)
Epirubicin	1.6	900
Idarubicin	0.3	150
Mitoxantrone	0.25	140

CV, cardiovascular.

SPECIAL CONSIDERATIONS

VENOUS THROMBOEMBOLISM (VTE) PREVENTION AND MANAGEMENT

- VTE—including deep vein thrombosis and pulmonary embolism—is a common cause of cancer-related morbidity and mortality. Cancer increases VTE risk 4- to 7-fold, affecting up to 15% of patients each year.
- Hospitalized patients with active cancer and those undergoing major cancer surgery should receive VTE prophylaxis with **unfractionated heparin** or **low-molecular-weight heparin** (LMWH), unless bleeding or other contraindications exist. Continue prophylaxis for 7–10 days post-op or up to 4 weeks after high-risk abdominal or pelvic surgery.
- Use the Khorana model to assess VTE risk in ambulatory patients receiving chemotherapy (see NCCN guidelines for score calculation). Offer prophylaxis with apixaban, rivaroxaban, or LMWH for up to 6 months or longer if high risk persists.
- In multiple myeloma patients on immunomodulatory agents, use the SAVED and IMPEDE VTE scores to determine prophylaxis needs (see NCCN

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guidelines for score algorithms).

- Treat VTE for at least 3–6 months or for as long as cancer is active or anticancer therapy continues. Refer to the NCCN and ASCO guidelines for drug dosing recommendations, including adjustments for thrombocytopenic patients.

PAIN MANAGEMENT

- Pain is a common symptom in patients with cancer, either acute or chronic, with various etiologies. Assess pain at initial evaluation, each subsequent contact, and when starting new therapy. Individualize treatment considering clinical circumstances and patient preferences, with the goal of maximizing function and quality of life. See Chapter 54 for more information on pain management.

LONG-TERM EFFECTS/SURVIVORSHIP

- Anticancer agents can cause long-term effects, including organ damage, reproductive issues, and immunologic or psychological effects. Advances in screening and early diagnosis of cancer as well as improvements in treatments have increased cancer survival, leading to the creation of multidisciplinary clinics to manage long-term complications.
- Survivors should be assessed for long-term physical and psychosocial effects such as organ dysfunction (eg, anthracycline-induced cardiotoxicity), pain or neuropathy, depression, cognitive issues, infertility, and secondary cancers.
- Consensus-based guidelines exist for long-term screening and follow-up, often specific to initial cancer type and treatment regimens, which is beyond the scope of this chapter.
- Cytotoxic chemotherapy, RT, and some oral inhibitors can increase the risk of secondary malignancies.

See Chapter 155, Supportive Care in Cancer, authored by Amber B. Clemmons and Alexandre Chan, for a more detailed discussion of this topic.

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